1 Introduction

The first British guidelines on asthma management in adults were published in the *British Medical Journal* in 1990 after a joint initiative between the British Thoracic Society (BTS), the Royal College of Physicians of London, the King's Fund Centre, and the National Asthma Campaign.[1 2] These were updated in 1993 with the addition of guidelines on childhood asthma[3] and further updated in 1995.[4] The Scottish Intercollegiate Guidelines Network (SIGN) published its first asthma guideline in 1996[5] and has subsequently published on primary care management of asthma in 1998[6] and management of acute asthma in 1999.[7]

Both the BTS and SIGN have recognised the need to update their asthma guidelines, using evidence-based methodology, to cover all aspects of asthma care. It was agreed that the two organisations should jointly produce a comprehensive new guideline, the process being further strengthened by collaboration with the National Asthma Campaign, the Royal College of Physicians of London, the Royal College of Paediatrics and Child Health, General Practice Airways group, and the British Association of Accident & Emergency Medicine. The outcome of these efforts is this new British Guideline on the Management of Asthma.

The new guideline has been developed using SIGN methodology, adapted for UK-wide development.[8] Further details about SIGN and the guideline methodology are contained in *SIGN 50; A Guideline Developer's Handbook* available at www.sign.ac.uk. Initial literature searches based on key questions produced over 15,000 abstracts and all relevant published papers up to the end of September 2001 have been considered. The work was divided between nine different multidisciplinary Evidence Review Groups whose members in combination encompass all relevant professional groups (see section 14.2). The guideline was discussed at an open meeting in Edinburgh in October 2001 and has also been formally reviewed by an extensive panel of peer reviewers (see section 14.4.2).

The levels of evidence and grades of recommendation used in this guideline are detailed in table 1.[9] Users should note that the grade of recommendation relates to the strength of the evidence and not necessarily to the clinical importance of the recommendation. Where there are only low grade recommendations in important clinical areas, this should be seen as a stimulus to further rigorous research.

The aim of the guideline is to provide comprehensive advice on asthma management for patients of all ages in both primary and secondary care that will be of use to all health professionals involved in the care of people with asthma.

Table 1 Key to evidence statements and grades of recommendations

LEVELS OF EVIDENCE

1^{++}	High quality meta-analyses, systematic reviews of randomised controlled trials (RCTs), or RCTs with a very low risk of bias
1^{+}	Well-conducted meta-analyses, systematic reviews, or RCTs with a low risk of bias
1^{-}	Meta-analyses, systematic reviews, or RCTs with a high risk of bias
2^{++}	High quality systematic reviews of case control or cohort studies High quality case control or cohort studies with a very low risk of confounding or bias and a high probability that the relationship is causal
2^{+}	Well conducted case control or cohort studies with a low risk of confounding or bias and a moderate probability that the relationship is causal
2^{-}	Case control or cohort studies with a high risk of confounding or bias and a significant risk that the relationship is not causal
3	Non-analytical studies, e.g. case reports, case series
4	Expert opinion

GRADES OF RECOMMENDATION

A

At least one meta-analysis, systematic review, or RCT rated as 1^{++}, and directly applicable to the target population; *or*

A body of evidence consisting principally of studies rated as 1^{+}, directly applicable to the target population, and demonstrating overall consistency of results

B

A body of evidence including studies rated as 2^{++}, directly applicable to the target population, and demonstrating overall consistency of results*; or*

Extrapolated evidence from studies rated as 1^{++} or 1^{+}

C

A body of evidence including studies rated as 2^{+}, directly applicable to the target population and demonstrating overall consistency of results*; or*

Extrapolated evidence from studies rated as 2^{++}

D

Evidence level 3 or 4*; or*

Extrapolated evidence from studies rated as 2^{+}

GOOD PRACTICE POINTS

✔ Recommended best practice based on the clinical experience of the guideline development group

2 Diagnosis & natural history

The diagnosis of asthma is a clinical one; there is no confirmatory diagnostic blood test, radiographic or histopathological investigation. In some people the diagnosis can be corroborated by suggestive changes in lung function tests.

The clinical diagnosis of asthma is not always simple (see fig 1) and the absence of an agreed definition of the disease is a problem, with many descriptions existing.[10] The International Consensus Report describes asthma as *"a chronic inflammatory disorder of the airways …. in susceptible individuals, inflammatory symptoms are usually associated with widespread but variable airflow obstruction and an increase in airway response to a variety of stimuli. Obstruction is often reversible, either spontaneously or with treatment"*.[11]

2.1 DIAGNOSIS OF ASTHMA IN ADULTS

Some of the symptoms of asthma are shared with diseases of other systems. Even when the symptom of breathlessness is thought to be due to lung disease, there are numerous relatively common lung diseases and differentiation of an airway disorder needs to be made from both infections, and pulmonary thromboembolic disease and restrictive lung disorders. Features of an airway disorder such as cough, wheeze and breathlessness should be corroborated where possible by measurement of airflow limitation. They may be due either to a localised airway obstruction (e.g. tumour, foreign body, vocal cord dysfunction or post tracheostomy stenosis), or to a generalised problem (such as asthma, chronic obstructive airways disease (COPD), bronchiectasis, cystic fibrosis or obliterative bronchiolitis).

2.1.1 SYMPTOMS OF ASTHMA

To avoid misdiagnosis it is essential to remember that people with asthma may suffer from a variety of symptoms, none of which is specific for asthma:

- wheeze
- shortness of breath
- chest tightness
- cough.

The hallmark of asthma is that these symptoms tend to be:

- variable
- intermittent
- worse at night
- provoked by triggers including exercise.

When cough is the predominant symptom without wheeze, this is often referred to as cough variant asthma.

2.1.2 SIGNS OF ASTHMA

During exacerbations, the patient will often have wheeze and reduced lung function, either reduced peak flow or an obstructive pattern on spirometry. The presence of wheeze (usually diffuse, polyphonic, bilateral and particularly expiratory) is a cardinal sign of asthma and, if present, should be documented in clinical notes. Outside acute episodes, there may be no objective signs of asthma (see section 2.1.4). Patients who present with chronic asthma may have signs of hyperinflation with or without wheeze.

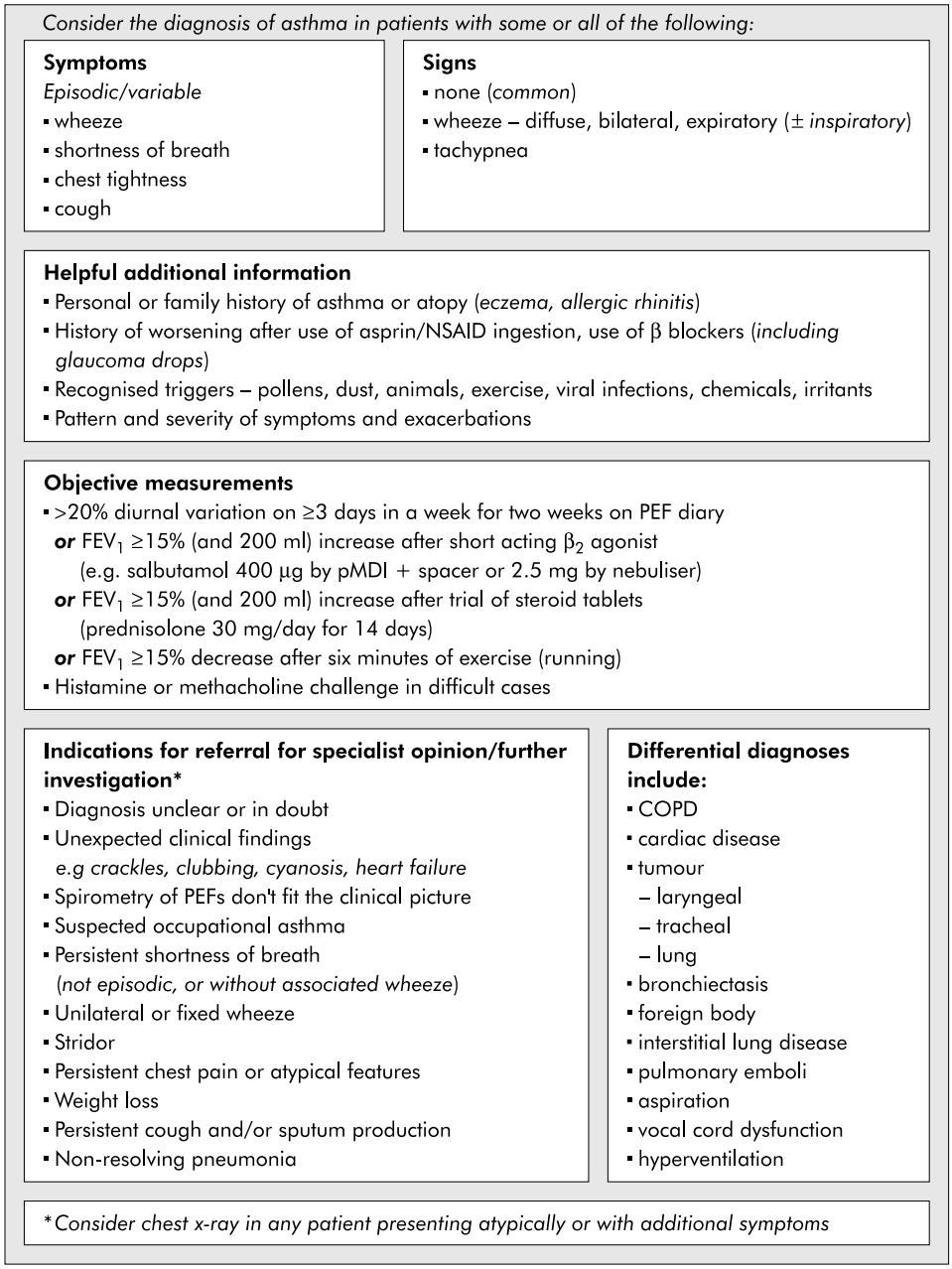
✔ Record the presence of wheeze in the patient's notes.

2.1.3 ADDITIONAL INFORMATION

Additional information which may contribute towards a clinical suspicion of asthma includes:

- personal or family history of asthma or other atopic condition (eczema, allergic rhinitis)
- worsening of symptoms after exposure to recognised triggers such as pollens, dust, feathered or furry animals, exercise, viral infections, chemicals, and environmental tobacco smoke
- worsening of symptoms after taking aspirin/non-steroidal anti-inflammatory medication or use of β blockers.

Figure 1 Diagnosis of asthma in adults



2.1.4 OBJECTIVE TESTS

Obstructive airways disease produces a decrease in peak expiratory flow (PEF) and forced expiratory volume in one second (FEV_1). One or both of these should be measured, but may be normal if the measurement is made between episodes of bronchospasm. If they are repeatedly normal in the presence of symptoms, then a diagnosis of asthma must be in doubt.

Variability of PEF and FEV_1, either spontaneously over time or in response to therapy is a characteristic feature of asthma. Although the normal level of diurnal variability is open to question, sequential measurement of PEF may be useful in the diagnosis of asthma. Calculating variability may be done in one of several ways and the method used should always be stated. A 20% or greater variability in amplitude % best (see box 1) with a minimum change of at least 60 l/min, ideally for three days in a week for two weeks seen over a period of time, is highly suggestive of asthma.[12–18]

Many patients with asthma will demonstrate variability below 20%, making this a reasonably specific, but insensitive diagnostic test. That is, marked variability of peak flow and easily demonstrated reversibility confirms a diagnosis of asthma, but smaller changes do not necessarily exclude the diagnosis.

Box 1 Diagnosis of asthma using PEF

Diagnosis of asthma using PEF

amplitude % best = (highest – lowest) / highest × 100
Highest PEF = 400 l/min
Lowest PEF = 300 l/min
Amplitude = 400 l/min – 300 l/min = 100 l/min
Percentage PEF variability = (400 – 300)/400 × 100 = 25%

Alternative methods for measuring variable airflow limitation are:

- an increase after inhalation of a short acting β_2 agonist (e.g. salbutamol 400 μg by metered dose inhaler (pMDI) + spacer or 2.5 mg by nebuliser)
- an increase after a trial of steroid tablets (prednisolone 30 mg/day for 14 days)
- a decrease after six minutes of exercise, e.g. running. Take a resting measurement, ask the patient to exercise for six minutes, take a further reading and then every 10 minutes for 30 minutes. As this procedure may rarely induce significant asthma, facilities for immediate treatment should be available.

✔ Objective tests should be used to try to confirm a diagnosis of asthma before long term therapy is started.

Each of the above methods can be used, measuring either PEF (look for a 20% change from baseline and at least 60 l/min) or FEV_1 (15% change and at least 200 ml).[19]

Increased bronchial responsiveness demonstrated by methacholine or histamine challenge is associated with symptomatic asthma, but is also common in the general population and in patients with COPD. However, failure to demonstrate hyperresponsiveness in an untreated person with suspected asthma should prompt reconsideration of the diagnosis.

2.1.5 OTHER TESTS

Lung function tests may show changes suggestive of an alternative lung disease. For example, COPD may be suspected in the presence of obstructive spirometry, reduced diffusing capacity (CO uptake) and pressure dependent airway collapse on flow volume curves, but these changes are not diagnostic and do not exclude asthma, which may anyway coexist with other conditions.

✔
- Failure to respond to asthma treatment should prompt a search for an alternative, or additional, diagnosis.
- Perform chest x-rays in all patients with atypical symptoms.

2.2 DIAGNOSIS OF ASTHMA IN CHILDREN

A definitive diagnosis of asthma can be difficult to obtain in young children (see fig 2). It is often not possible to measure airway function in order to confirm the presence of variable airway obstruction.

✔ Asthma should be suspected in any child with wheezing, ideally heard by a health professional on auscultation, and distinguished from upper airway noises.

In schoolchildren, bronchodilator responsiveness, PEF variability or tests of bronchial hyperreactivity may be used to confirm the diagnosis, with the same reservations as in adults (see section 2.1.4).

Allergy tests may be helpful in seeking causal factors, and in making a general diagnosis of atopy. The presence of allergy is not essential to the diagnosis of asthma, but its absence in a school child with symptoms suggestive of asthma should prompt consideration of alternative diagnoses.

D

Base the diagnosis of asthma in children on:

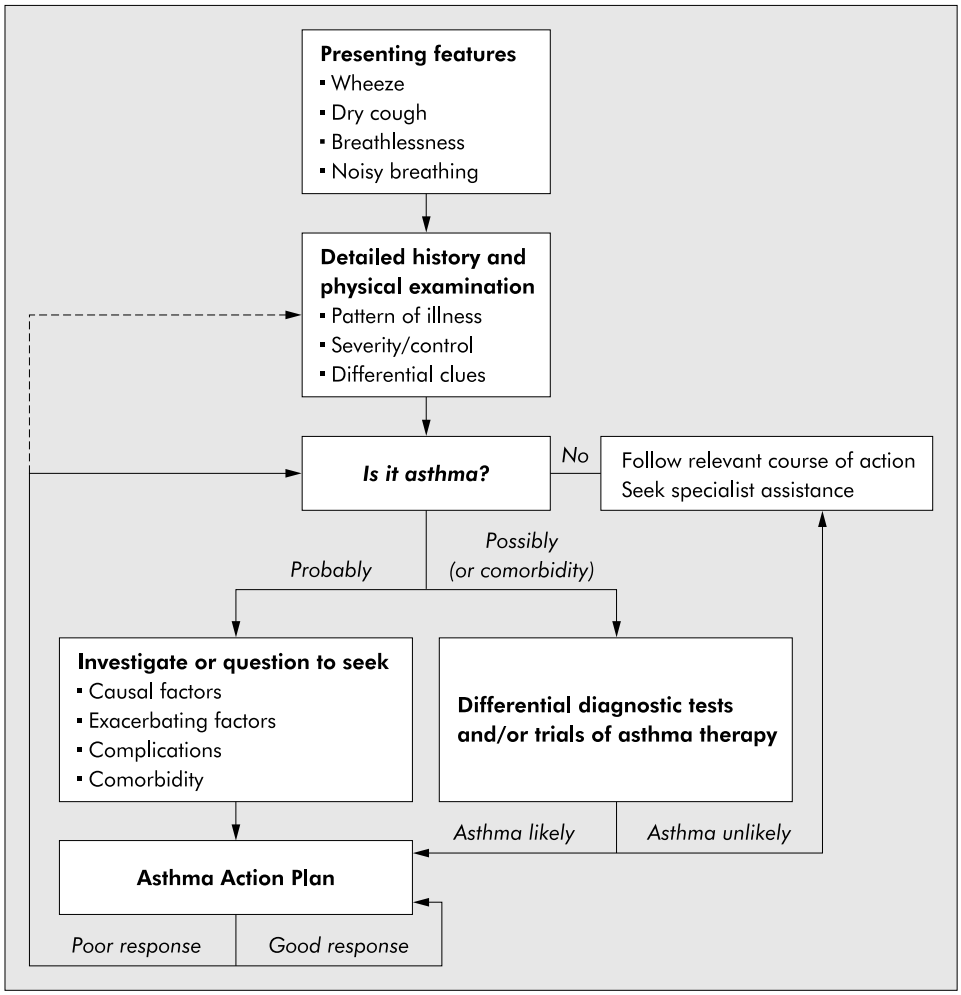
- **the presence of key features and careful consideration of alternative diagnoses** (see table 2)
- **assessment of the response to trials of treatment, and ongoing assessment**
- **repeated reassessment of the child, questioning the diagnosis if management is ineffective.**

✔ Record the criteria on which the diagnosis has been made.

Table 2 Clues to alternative diagnoses in wheezy children (features not commonly found in asthma)

Clinical clue	Possible diagnosis
Perinatal and family history	
Symptoms present from birth or perinatal lung problem	Cystic fibrosis; chronic lung disease; ciliary dyskinesia; developmental anomaly
Family history of unusual chest disease	Cystic fibrosis; developmental anomaly; neuromuscular disorder
Severe upper respiratory tract disease	Defect of host defence
Symptoms and signs	
Persistent wet cough	Cystic fibrosis; recurrent aspiration; host defence disorder
Excessive vomiting or posseting	Reflux (± aspiration)
Dysphagia	Swallowing problems (± aspiration)
Abnormal voice or cry	Laryngeal problem
Focal signs in the chest	Developmental disease; postviral syndrome; bronchiectasis; tuberculosis
Inspiratory stridor as well as wheeze	Central airway or laryngeal disorder
Failure to thrive	Cystic fibrosis; host defence defect; gastro-oesophageal reflux
Investigations	
Focal or persistent radiological changes	Developmental disorder; postinfective disorder; recurrent aspiration; inhaled foreign body; bronchiectasis; tuberculosis

Figure 2 Diagnosis of asthma in children



Box 2 Indications for referral

Indications for referral for specialist opinion/further investigation

- Diagnosis unclear or in doubt
- Symptoms present from birth or perinatal lung problem
- Excessive vomiting or posseting
- Severe upper respiratory tract infection
- Persistent wet cough
- Family history of unusual chest disease
- Failure to thrive
- Unexpected clinical findings
  e.g. focal signs in the chest, abnormal voice or cry, dysphagia, inspiratory stridor
- Failure to respond to conventional treatment
  (*particularly inhaled corticosteroids above 400 µg /day or frequent use of steroid tablets)*
- Parental anxiety or need for reassurance

2.3 PROGNOSIS OF CHILDHOOD ASTHMA

Therapeutic decisions, particularly the introduction of prophylactic treatments may be influenced not only by the presence of persistent symptoms but by current understanding of the pathophysiology and the natural history of the disease.

If the factors associated with resolution and persistence of asthma presenting in childhood were not taken into account and every child presenting with wheeze was treated prophylactically, half of all children would be treated.

The major identifiable risk factors contributing to both the expression and persistence of asthma are considered below.

2.3.1 FAMILY HISTORY OF ATOPY

A family history of atopy is the most important clearly defined risk factor for atopy in children. Asthma is linked to both parental and sibling atopy. The strongest association is with maternal atopy. A maternal history of asthma and/or rhinitis is a significant risk factor for late childhood onset asthma and recurrent wheezing throughout childhood. The association of persistence of symptoms with maternal asthma and rhinitis weakens during the transition to adulthood.[20–33]

2.3.2 CO-EXISTENCE OF ATOPIC DISEASE

Markers of allergic disease at presentation, (including skin prick tests, eosinophil counts and peripheral blood markers), are related to severity of current asthma and persistence through childhood, but as yet have not been shown to be related to the outcome of respiratory symptoms and their severity in adulthood.[20 22 27 31 33-42]

2.3.3 EFFECT OF SEX

Male sex is a risk factor for asthma in prepubertal children and female sex is a risk factor for persistence of asthma in the transition from childhood to adulthood. Male children with asthma are more likely to "grow out" of their asthma in the transition to adulthood.[21 22 24 26 33 35 42–52]

2.3.4 BRONCHIOLITIS IN INFANCY

Viral associated wheeze in infancy is often followed by wheeze in early childhood. This association weakens with advancing age and by 35–40 years ventilatory function and bronchial reactivity is similar to those who had no symptoms as children.[22 30 32 38 49 53–58]

2.3.5 PARENTAL SMOKING

Maternal smoking is associated with significantly higher prevalence of wheezing illness in early childhood. However, there is no identifiable association between parental smoking and respiratory symptoms in adult life. Reducing the prevalence of smoking in the adult population, and particularly in women of childbearing age, would significantly reduce the prevalence of wheezing in young children.[20-24 26 27 32 36 59–61]

2.3.6 BIRTH WEIGHT AND PREMATURITY

Wheezing is more common in young children who were born prematurely. In adulthood there are no consistent relationships between asthma and birth weight.[21 30 59 60 62 63]

2.3.7 AGE AT PRESENTATION

The natural history of wheeze is dependent on the age at first presentation. The earlier the onset of wheeze, the better the prognosis. Available data from child cohorts show a "break point" at two years with the majority of those presenting before this age becoming asymptomatic by mid childhood (6–11 years). It must be remembered that coexistent atopy (see section 2.3.2) is a risk factor for persistence independent of age of presentation.[20 33 34 38 42 43 47 48 54 64–69]

2.3.8 SEVERITY AND FREQUENCY OF EPISODES

Increased frequency and severity of wheezing episodes in childhood are associated with recurrent wheeze into adulthood.[20 28 33 34 39 41 43 46 52 68 70 71]

2.3.9 LUNG FUNCTION MEASUREMENTS

There is a relationship between the level of pulmonary function in childhood and in adulthood. Persistent reduction in baseline airway function and increased airway responsiveness is associated with continuation of symptoms into adulthood.[20 22 40 43 52 53 70–73]

3 Non-pharmacological management

There is increasing interest in factors which, if avoided, might facilitate the management of asthma, reducing the requirement for pharmacotherapy; and which may have the potential to modify fundamental causes of asthma. However, evidence has been difficult to obtain for many approaches and more studies are required.

This section distinguishes:

- primary prophylaxis: interventions made before any evidence of disease
- secondary prophylaxis: interventions made after the onset of disease to reduce its impact.

The distinction is made as factors that induce the disease in the first place are not necessarily the same as those that incite a pre-existing problem.

3.1 PRIMARY PROPHYLAXIS

Primary prophylaxis is employed before there is any evidence of disease in an attempt to prevent its onset. A number of potential strategies are discussed below.

3.1.1 ALLERGEN AVOIDANCE

There is a strong correlation between allergic sensitisation to common aeroallergens and the subsequent development of asthma. There is also a strong association between allergen exposure in early life and sensitisation to these allergens, although it has not been possible to demonstrate an association between allergen exposure and the development of asthma.[74]

The majority of allergen avoidance studies focus on dietary manipulation to prevent atopic eczema and have paid little attention to aeroallergen avoidance. Two trials in progress are investigating the consequences of introducing house dust mite reduction in early pregnancy, and are following up the children born to the participating mothers. Although accurate asthma phenotyping is not possible in infancy, outcomes at one year of age indicate a modest but significant reduction in wheezing illnesses.[75 76]

Allergen avoidance after birth has been studied in a number of controlled (but not double blind) trials. There appears to be a transient reduction in the prevalence of atopic eczema in the first two years of life but no evidence of sustained benefit in relation to asthma.[77 78] A number of epidemiological studies suggest that close contact with a cat or dog in very early infancy reduces subsequent prevalence of allergy and asthma. This may be a consequence of high allergen exposure inducing tolerance.[79–81]

No recommendations on prenatal or postnatal allergen avoidance can be made in relation to primary prevention of asthma.

3.1.2 BREAST FEEDING

A systematic review and meta-analysis involving 8183 subjects followed for a mean of four years revealed a significant protective effect of breast feeding against the development of asthma. The effect was greatest in children with a family history of atopy.[82] In contrast, a more recent study in 1246 patients found that breast feeding was associated with a reduced risk of infant wheeze, but also with an increased risk of asthma at six years.[83] | I+

A **Breast feeding should be encouraged and its benefits include a protective effect in relation to early life wheezing.**

3.1.3 MODIFIED INFANT MILK FORMULAE

Trials of modified milk formulae using partial and extensive hydrolysates of whey or casein or soy formulae compared with conventional formulae have not shown any consistent significant long term benefits in relation to asthma. Variation in study design, intervention used, co-interventions and outcome definition make meta-analysis problematical.[84]

3.1.4 OTHER DIETARY MODIFICATIONS

Limited epidemiological evidence suggests that fish oil consumption may protect against asthma in childhood.[85] Trials of lipid supplementation during pregnancy and postnatally to prevent atopic disease are in progress.

3.1.5 MICROBIAL EXPOSURE

The "hygiene hypothesis" suggests that early exposure to microbial products will switch off allergic responses preventing allergic diseases such as asthma.[86] Epidemiological studies comparing large populations who have or have not had such exposures support the hypothesis.[87] A double blind placebo trial of the probiotic, Lactobacillus CG, reported a reduced incidence of atopic eczema but no effect on IgE antibody sensitisation. Small sample size and early outcome age limit the interpretation of this study.[88] In the absence of good quality intervention studies, no recommendation can be made at present.

3.1.6 IMMUNOTHERAPY AND PRIMARY PREVENTION

Three observational studies, in over 8000 patients, have shown that immunotherapy in individuals with a single allergy reduces the numbers subsequently developing new allergies over a three to four year follow up compared with contemporaneous untreated controls.[89–91] No double blind placebo controlled trials of immunotherapy as primary prevention have been conducted, and at present immunotherapy cannot be recommended for primary prevention. Preliminary results from an ongoing parallel group study using contemporaneous untreated children as the control group for pollen immunotherapy in children with allergic rhinitis suggest a lower rate of onset of asthma in the treated group.[92]

3.1.7 AVOIDING POLLUTANTS

No evidence was found to support a link between exposure to environmental tobacco smoke and other air pollutants and the induction of atopic asthma.

An early meta-analysis suggested an association between gas cooking and respiratory illness[93] but this has not been borne out in larger studies.[94 95]

Increased risk of infant wheeze is associated with smoking during pregnancy and maternal postnatal smoking.[96] Pregnancy smoking affects an infant's airway function, increasing susceptibility to wheeze.[97–101] There are many other adverse effects on the young child of such exposures. 2^{++}

B **Parents and parents-to-be who smoke should be advised of the many adverse effects of smoking on their children, including increased wheezing in infancy, and be offered appropriate support to stop smoking.**

3.1.8 PHARMACOTHERAPY

For completeness, this section on the primary prevention of asthma should mention pharmacological trials of treatments designed to prevent onset of the disease. Children given ketotifen (206 infants, in two trials) showed significantly less asthma at one and three year follow up compared with those receiving placebo.[102 103] In the third study, using cetirizine, 18 months' treatment had no effect in the intention to treat population but significantly reduced asthma in children with atopic dermatitis sensitised to either grass pollen or house dust mite. Cetirizine had additional benefits for atopic dermatitis alone and reduced the frequency of urticaria.[104]

3.2 SECONDARY NON-PHARMACOLOGICAL PROPHYLAXIS

3.2.1 ALLERGEN AVOIDANCE

Allergen avoidance measures may be helpful in reducing the severity of existing disease. Increasing allergen exposure in sensitised individuals is associated with an increase in asthma symptoms, bronchial reactivity and deterioration in lung function.[105–107]

Treatment requirements, hospital attendance and respiratory arrest are associated with increased exposure to high concentrations of indoor allergens.[108]

Threshold concentrations of allergens that can be regarded as risk factors for acute attacks include:

- 10 μg/g dust of group 1 mite allergen[109]
- 8 μg/g dust of Fel d 1, the major cat allergen[109]
- 10 μg/g dust of Can f 1, the major dog allergen[109]
- 8 μg/g dust of cockroach allergen.[110]

Evidence that reducing allergen exposure can reduce morbidity and mortality is tenuous. In uncontrolled studies, children and adults have both shown benefit from exposure to a very low allergen environment. However, the benefits in such circumstances cannot be necessarily attributed to the allergen avoidance.[111–113]

3.2.2 HOUSE DUST MITE CONTROL MEASURES

There have been two Cochrane reviews on house dust mite control measures and the management of asthma.[114 115] The first concluded that current chemical and physical methods were ineffective and could not be recommended as prophylactic treatment for asthma patients with sensitivity to house dust mites. An amendment concluded that physical reduction methods may reduce asthma symptoms.[115]

The reviewed studies used various chemical, physical or combinations of methods to reduce mite exposure. The combined meta-analysis showed no difference in improvement in asthma between patients in experimental groups compared with controls. There was heterogeneity between studies with regard to intervention, and in some studies intervention allocation was not adequately concealed.[115]

Larger and more carefully controlled studies are required to demonstrate any clear benefit from house dust mite avoidance. At present, this does not appear to be a cost-effective method of achieving benefit.

✔

In committed families with evidence of house dust mite allergy and who wish to try mite avoidance, the following are recommended:[116]

- complete barrier bed covering systems
- removal of carpets
- removal of soft toys from bed
- high temperature washing of bed linen
- acaricides to soft furnishings
- dehumidification.

3.2.3 OTHER ALLERGENS

Animal allergens, particularly cat and dog, are a potent cause of asthma symptoms. Observational studies have not found that removing a pet from a home improves asthma control.[117] In a study in adults with cat sensitivity, randomisation to either bedroom air cleaner and covers for bedding or no active intervention with restriction of cats away from the bedroom, resulted in no differences between groups with regard to symptoms, peak flow, lung function or bronchial reactivity.[118] Alternatively, there is a suggestion that maintaining a high exposure to cat allergen in the domestic environment might actually induce some degree of tolerance.[81] Many experts still feel that removal of pets from the home of individuals with asthma who also have an allergy to that pet should be recommended.

Cockroach allergy is not a common problem in the UK. There is no conclusive evidence regarding the impact of cockroach allergen reduction on asthma symptoms.[119]

Although fungal exposure has been strongly associated with hospitalisation and increased mortality in asthma, to date no controlled trials have addressed fungal exposure reduction and asthma.[120]

3.3 ENVIRONMENTAL FACTORS

3.3.1 SMOKING

The association between passive smoking and respiratory health has been extensively reviewed.[121] There is a direct causal relationship between parental smoking and lower respiratory illness in children up to three years of age. Infants whose mothers smoke are four times more likely to develop wheezing illnesses in the first year of life.[97]

The independent contributions of prenatal and postnatal maternal smoking to the development of asthma in children are difficult to distinguish.[121] Maternal pregnancy smoking has been shown to have an adverse influence on lung development.[97–99] There is little evidence that maternal pregnancy smoking has an effect on allergic sensitisation.[121]

Exposure to tobacco smoke in the home contributes to the severity of childhood asthma. A US Institute of Medicine review identified a causal relationship between environmental tobacco smoke (ETS) exposure and exacerbations of asthma in pre-school children. Average exposure is associated with a 30% increased risk of symptoms.[122] One small study suggests that by stopping smoking, parents decrease the severity of asthma in their children.[123]

B **Parents who smoke should be advised about the dangers for themselves and their children and offered appropriate support to stop smoking.**

Starting smoking as a teenager increases the risk of persisting asthma. Only one study was identified that examined the incidence of asthma related to taking up smoking. This showed a relative risk of 2.1 for the development of asthma over six years in 14 year old children who have started to smoke.[124]

No studies were identified that directly related to the question of whether smoking affects asthma severity. One controlled cohort study suggested that exposure to passive smoke at home delayed recovery from an acute asthma attack.[125] Studies of interventions designed to reduce environmental tobacco smoke exposure in the home have been largely ineffective in reducing the degree of exposure and none were designed with primarily clinical (as opposed to smoking) outcomes.[126 127] In one observational study, giving up smoking in adults was associated with improved severity of asthma scores.[128]

✔ Smoking cessation should be encouraged as it is good for general health and may decrease asthma severity.

3.3.2 AIR POLLUTION

There is evidence that changing from a high particulate sulphur dioxide (coal burning) environment to a low sulphur dioxide/high diesel particulate environment increases the incidence of asthma and atopy.[129 130] In the UK, asthma is more prevalent in 12–14 year olds in non-metropolitan rather than metropolitan areas.[131] However, many differences between environments might explain the variation in asthma and allergy risk. There is some laboratory evidence that various pollutants can enhance the response of patients with asthma to allergens,[132 133] but there is no firm epidemiological evidence that this has occurred in the UK or elsewhere.[134]

Time series studies suggest that air pollution may provoke acute asthma attacks or aggravate existing chronic asthma, although the effects are minimal in comparison with factors such as infection. The short term fluctuations in levels of air pollution currently encountered in the UK may be responsible for small changes in numbers of hospital admissions and A&E attendances for asthma.[134]

No evidence was identified regarding asthma and indoor air pollutants, such as volatile organic compounds, formaldehyde or nitrogen oxides.[135 136] Further research in this area is required.

3.4 COMPLEMENTARY AND ALTERNATIVE MEDICINE

3.4.1 HERBAL AND TRADITIONAL CHINESE MEDICINE

Currently available evidence does not allow any firm judgement to be made on herbal remedies in general or individual preparations in particular. Seventeen trials were identified, but the combined results are inconclusive. Nine of the 17 trials reported some improvement in lung function, but it is not clear that the results reported would be generalisable to a UK population.[137]

3.4.2 ACUPUNCTURE

A Cochrane review[138] of 21 trials raised many methodological concerns. Only seven trials (174 patients) achieved randomisation to active (i.e. recognised in traditional Chinese medicine to be of benefit in asthma) or sham acupuncture (i.e. points with no recognised activity) for the treatment of persistent or chronic asthma. Blinding was a common problem, and only achieved for those making the observations. The difficulty in making sham acupuncture convincing and part of the holistic approach of traditional Chinese medicine was emphasised. There was wide inconsistency in methodology. Acute trials show that acupuncture has a beneficial effect, but this is less in magnitude than that achieved by inhaled bronchodilators or cromones. Demonstrating that this effect can be transferred to persistent asthma using regular treatment was achieved in one RCT reported in the Cochrane review.

The Cochrane review found no evidence for a clinically valuable benefit from acupuncture, with no statistically significant improvement in lung function being demonstrated. More rigorous research methodology and attention to outcomes other than lung function are required.

3.4.3 AIR IONISERS

Ionisers are widely advertised and marketed as being of benefit to patients with asthma, however there is no evidence that they are of value in ameliorating the symptoms of asthma or improving lung function. They do reduce mite allergen levels in the room in which they are used, and could be incorporated into a co-ordinated allergen avoidance programme, but this has not been formally tested.

One study has raised concerns that ionisation may produce an increase in nocturnal cough.[139]

✔ The use of ionisers cannot be encouraged, as there is no evidence of benefit and a suggestion of adverse effect.

3.4.4 HOMEOPATHY

A Cochrane Review[140] identified only three methodologically sound RCTs. In the first trial (24 patients), homeopathy improved symptom scores and forced vital capacity (FVC) but had no effect on FEV_1 or bronchial reactivity. The second study demonstrated improvements in both active and placebo groups. The third, poorly reported, trial demonstrated an increase in lung function in patients receiving the active preparation.

There is insufficient information regarding the value of homeopathy in the treatment of asthma. Large well designed trials using defined remedies and a spectrum of patients are warranted.

3.4.5 HYPNOSIS

Studies of hypnosis in patients with asthma are generally poorly controlled and patient characteristics and outcome measures vary enormously. The conclusions from a critical review[141] were that hypnosis may be effective for asthma with the biggest effect in susceptible subjects, but more randomised and appropriately controlled studies are required.

3.4.6 MANUAL THERAPY INCLUDING MASSAGE AND SPINAL MANIPULATION

A Cochrane review identified four relevant RCTs.[142] The two trials of chiropractice

suggest that there is no place for this modality of treatment in the management of asthma. No conclusions can be drawn on massage therapy.

3.4.7 PHYSICAL EXERCISE TRAINING

A Cochrane review[143] has shown no effect of physical training on PEF, FEV_1, FVC or VE_{max}. However oxygen consumption, maximum heart rate, and work capacity all increased significantly. Most studies discussed the potential problems of exercise-induced asthma, but none made any observations on this phenomenon. As physical training improves indices of cardiopulmonary efficiency, it should be seen as part of a general approach to improving lifestyle and rehabilitation in asthma, with appropriate precautions advised about exercise-induced asthma (see section 4.7.2).

3.4.8 BREATHING EXERCISES INCLUDING YOGA AND BUTEYKO

The underlying principle of yoga and Buteyko is to reduce hyperventilation by lowering respiratory frequency. A Cochrane review[144] found no change in routine measures of lung function. Two studies reported a reduction in use of medication, and two a reduced frequency of attacks. At present it is not possible to make an evidence-based recommendation about breathing exercises for asthma.

3.4.9 FAMILY THERAPY

A Cochrane review identified two trials, in 55 children, showing that family therapy may be a useful adjunct to medication in children with asthma.[145] Small study size limits the recommendations.

✔ In difficult childhood asthma, there may be a role for family therapy as an adjunct to pharmacotherapy.

3.5 DIETARY MANIPULATION

3.5.1 MINERALS

Low magnesium intakes have been associated with higher prevalence of asthma. An intervention study of magnesium supplementation has suggested a reduced rate of bronchial hyperresponsiveness and wheeze.[146] Studies of sodium[147] and antioxidant supplements such as selenium and vitamin C[148] have produced little or no evidence of benefit amongst patients with asthma.

3.5.2 FISH OILS AND FATTY ACIDS

In vitro studies suggest that supplementing diet with the omega n-3 fatty acids found predominantly in fish oils might reduce the inflammation associated with asthma.[149] Controlled clinical studies in small numbers have on the whole been negative, with a Cochrane review concluding that there was little evidence to recommend fish oil supplements in asthma.[150]

3.5.3 WEIGHT REDUCTION IN OBESE PATIENTS WITH ASTHMA

A small randomised parallel group study has shown improved asthma control following weight reduction in obese patients with asthma.[151] | 2^+

C **Weight reduction is recommended in obese patients with asthma to improve asthma control.**

3.6 GASTRO-OESOPHAGEAL REFLUX IN ASTHMA

A Cochrane review of 12 double blind controlled trials found that treatment of gastro-oesophageal reflux had no benefit on asthma symptoms or lung function, when both conditions were present. Reduction in dry cough was observed, although this was probably not due to asthma.[152]

B **Gastro-oesophageal reflux should be treated if present but this will generally have no impact on asthma control.**

3.7 HIGH ALTITUDE AND SPELEOTHERAPY

Speleotherapy involves the use of subterranean environments as a therapeutic measure. A Cochrane review of the available evidence does not permit reliable conclusions to be drawn, although in two out of three studies, a total of 124 asthmatic children showed some short term benefit.[153] Randomised controlled trials with longer term follow up are required.

Moving children to high altitude environments with low allergen and pollutant exposure has been reported to be associated with clinical improvements,[111 113] but there are no published controlled trials and no long term follow up data available.

3.8 IMMUNOTHERAPY

Trials of allergen-specific immunotherapy (hyposensitisation or desensitisation) by subcutaneous injection of increasing doses of allergens have been systematically reviewed.[154–156] Three reviews have demonstrated consistent beneficial effects of the treatment compared with placebos. Allergens used in the studies included mites, pollen, animal danders, and moulds.

However, there are as yet no properly controlled studies making direct comparisons between conventional asthma pharmacotherapy and allergen immunotherapy. The preparation of materials for immunotherapy, dose frequency and duration of therapy has not been optimised; and the risk benefits compared with pharmacotherapy require careful consideration.

Immunotherapy may reduce asthma symptoms and use of asthma medications, but the size of benefit compared with other therapies is not known. Further comparative studies are needed.